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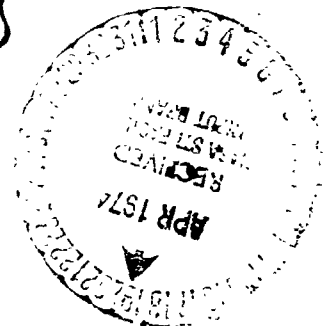
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VENOUS RETURN CURVES OBTAINED FROM
GRADED SERIES OF VALSALVA MANEUVERS

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ABSTRACT

We studied the effects of graded series of Valsalva-like maneuvers on venous return, measured transcutaneously in the jugular vein of an anesthetized dog, with the animal serving as its own control. At each of five different levels of central venous pressure we determined the airway pressure which just stopped venous return during each series of maneuvers. We found that, contrary to a previous hypothesis, this end-point airway pressure is not a good estimator of the animal's resting central venous pressure measured just prior to the simulated Valsalva maneuver. We found that the measured change in right atrial pressure during a Valsalva maneuver is less than the change in airway pressure during the same maneuver, instead of being equal, as we had expected. We constructed relative venous return curves from the data obtained during the graded series of Valsalva maneuvers, and estimated values of mean systemic pressure and venous return pressure from our relative venous return curves. We conclude that clinical application of our technique for estimating mean systemic pressure must await further evaluation studies in animals.

INDEX TERMS

central venous pressure; airway pressure; right atrial pressure; intrathoracic pressure; mean systemic pressure; relative venous return; venous return pressure; Doppler ultrasonic flowmeter; lung compliance; dog

The Subcommittee for Respiration of the International Union of Physiological Sciences Committee on Nomenclature defines Valsalva's maneuver as "expiratory effort with closed airway"(3). Clinically, the maneuver is usually performed against either a closed glottis or a mercury manometer with the glottis open. If the patient contracts his abdominal and thoracic expiratory muscles maximally, he may generate an intrathoracic pressure of up to 200 mm Hg (8). A more standardized experiment is for the physician to ask the subject to blow the mercury column to a given height (usually 40 mm Hg) and maintain it there for 10 to 15 seconds (28,7).

Most general physiology texts contain qualitative statements to the effect that venous return is diminished during a forced expiratory (Valsalva) maneuver. However, there appears to be some uncertainty concerning the exact mechanism causing this phenomena. Lambertsen (16) claims that elevated intrathoracic pressure collapses the central veins and thereby causes venous return to cease altogether. He states that following this collapse, blood accumulates in the periphery until extra-thoracic venous pressure overcomes the effects of abnormally high intrathoracic pressure and venous flow again begins. On the other hand, Sharpey-Schafer (28) does not mention the possibility of central venous collapse when he attributes the accumulation of blood in the peripheral veins to the increased intrathoracic pressure acting as an undefined "simple obstruction" to flow into the central veins.

Cessation of peripheral venous flow in humans during a Valsalva maneuver was first detected noninvasively by Rositano (25,27) using a transcutaneous Doppler ultrasonic flowmeter (31) with the probe over a vein in the antecubital fossa. Rositano's subjects sat or stood with their arms at heart level and elevated their intrathoracic pressure by exhaling through a constricted orifice. Webster (33,26) improved Rositano's technique by placing his subjects in the supine position, thus ensuring that the veins between the elbow and the heart were not collapsed prior to the maneuver. He also emphasized the possible application of Rositano's technique to the noninvasive measurement of resting central venous pressure (CVP). Webster assumed that when intrathoracic pressure (P_{it}) was elevated above the value of resting CVP, the central veins would collapse and venous return would cease. He instructed his subjects to generate a level of intrathoracic pressure which would just stop venous return as detected with the transcutaneous Doppler flowmeter. He reasoned that at the instant when venous return first stopped during a Valsalva maneuver, intrathoracic pressure would be equal to resting CVP and could be estimated by noting the airway (intrapulmonary) pressure (P_{ao}) at that instant and subtracting a correction factor to account for transpulmonary pressure (P_l). Ogden and Wells (23,34,35) have applied Rositano's technique to dogs with somewhat inconclusive results.

To quantitate and explain the effects of the Valsalva maneuver on venous return, we performed a series of experiments using an anesthetized dog as its own control. In designing these experiments we had the following purposes: 1) to determine if the level of airway pressure which just stops jugular venous flow during a Valsalva maneuver is a good estimator of resting central venous pressure measured just prior to the maneuver; 2) to elucidate the mechanism by which venous return is affected by changes in intrathoracic pressure; 3) to determine the relationship between airway pressure and CVP during a Valsalva maneuver and to determine if this relation is different from the one which holds when the lungs are inflated by external means; 4) to suggest or discount possible clinical applications of our measurements, such as determining the adequacy of circulating blood volume in the patient undergoing intravenous volume expansion.

METHODS

A 19.1 kg female mongrel dog was held NPO for 42 hr before the start of the experiment so as to ensure a low initial CVP (weight at beginning of experiment, 18.2 kg). The dog was anesthetized with 30 mg/kg of sodium pentobarbital (Nembutal), intubated with a cuffed endotracheal tube, and prepared as shown in Fig. 1. To prevent any spontaneous ventilatory activity from interfering with the experiment, the dog was paralyzed with a 10 mg loading dose and sustained with a 1 mg/min drip of succinylcholine chloride (Succostrin). The pressure-limited

ventilator (Bird Mark 7) was set to deliver 100% O_2 at a rate of 14 breaths/min with a peak pressure of 13 cm H_2O . The probe of the transcutaneous bidirectional Doppler ultrasonic blood flow velocity meter (Parks 806) was placed on the shaved neck surface over the right external jugular vein. The theoretical and practical characteristics of this instrument have been described in detail elsewhere (31,17,18,32,10,5), and our jugular venous flow velocity records compare favorably with other workers' indirect measures of superior vena caval flow (2,4). The reference port of the right atrial pressure transducer (Statham P23BB) and the zero of the CVP manometer scale were aligned to a level 61% of the thickest part of the chest above the back. This is the level of Guyton's physiologic reference point in the supine dog and corresponds anatomically to the opening of the tricuspid valve into the right ventricle (14). All measurements were made with the animal in the supine position, and the following variables were recorded on a strip-chart recorder (Brush 440): jugular venous flow velocity (U_{jv}), right atrial pressure (P_{ra}), and airway pressure (P_{ao}).

Valsalva maneuvers were simulated using a modification of the technique of Wells and Ogden (35): The 3-way valve shown in Fig. 1 was turned to open the dog's airway to the atmosphere, and the lungs were allowed to come to their equilibrium volume (functional residual capacity or FRC).¹ The airway was then closed, and airway pressure was increased in a stepwise manner by manually compressing the chest (and, in some maneuvers, the abdomen) at the cross-hatched regions in Fig. 1. Airway pressure was held at a

constant level for several seconds (range, 6 to 12 sec) and then released. We again connected the dog's airway to the ventilator for at least one minute before repeating the above procedure for a different value of airway pressure. We define a graded series of Valsalva maneuvers to be a consecutive repetition of the above experiment wherein each maneuver is to a greater (but always constant) level of airway pressure than the previous one in the series. In each series of simulated Valsalva maneuvers performed on our experimental animal, levels of airway pressure were generated in the above manner in increments of 5 cm H₂O from zero to a value which caused the jugular venous flow velocity to be zero for at least one heart cycle. We define this final level of airway pressure to be the value of the endpoint airway pressure ($P_{ao'_M}$) for the given series. At the dog's initial low CVP level we also performed a series of stepwise changes in airway pressure by inflating the dog's lungs (chest wall and abdomen not restrained) using a Hope bag resuscitator attached to the tee on the endotracheal tube in place of the Bird ventilator (Fig. 1).

In order to study venous return before, during, and after expansion of circulatory volume, we infused lactated Ringer's solution so as to raise the resting central venous pressure in several increments from its initial level of -0.6 cm H₂O to greater than 20 cm H₂O. At each level of CVP a graded series of Valsalva maneuvers was performed while CVP was held constant by manually adjusting the rate of infusion of the Ringer's solution. The five different levels of resting, pre-maneuver central venous pressure and the seven different experimental conditions are summarized in Table 1.

RESULTS

The records shown in Fig. 2 represent a typical study of a particular level of airway pressure generated by a single simulated Valsalva maneuver. In this particular maneuver venous return is decreased, but a higher level of airway pressure is needed to stop venous return for at least one heart cycle. Note that the recordings obtained allow the correlation of jugular venous flow velocity, right atrial pressure, and airway pressure at any instant during any given maneuver. Our analysis emphasizes two particular points in time: 1) just prior to each maneuver when the lungs are at FRC, since variables measured here are at their resting values for a particular CVP level; 2) at the instant when venous return reaches a minimum during the maneuver, since at this point in time a given level of airway pressure is exerting its maximum effect on venous return.

Records like those shown in Fig. 2 were obtained for all of the maneuvers performed in each of the graded series of Valsalva maneuvers described in the first six lines of Table 1. As discussed in the METHODS section, a value of end-point airway pressure (P_{ao_M}') was determined for each of the series A.I. through E.II. by noting the first value of P_{ao_M} in each graded series of maneuvers which corresponded to a U_{jv_M} with a value of zero. In series A.III. (lung inflations with the Hope bag), however, even an airway pressure of 17 cm H_2O had no appreciable effect on venous return; therefore, U_{jv_M} values were undefined, and no value of end-point airway pressure (P_{ao_M}') could be determined for that series. For comparison purposes we did define values of P_{ra_M} and P_{ao_M} for each

maneuver in series A.III. by measuring them 2 sec after the initial rise in airway pressure.

Our first result was obtained by comparing resting, pre-maneuver central venous pressure for each series with the value of end-point airway pressure for the same series. The pre-maneuver airway pressure (Pao_0) was sometimes slightly above atmospheric (due to the investigator's placing his hands on the chest just prior to the maneuver). We corrected for this small pressure shift using the equation,

$$\Delta Pao' = Pao'_M - Pao'_0 \quad (1)$$

where Pao'_0 is the pre-maneuver airway pressure for the maneuver which defines the end-point airway pressure (Pao'_M) in each series. We plotted this baseline-corrected end-point airway pressure ($\Delta Pao'$) against pre-maneuver CVP (Fig. 3) to show Result 1: In the anesthetized dog the level of airway pressure which just stops venous return during a Valsalva maneuver is not a good estimator of resting central venous pressure.

To help us understand the mechanism by which changes in intrathoracic pressure affect venous return, we followed a data analysis procedure which we developed for noninvasively measuring relative venous return in humans during graded series of Valsalva maneuvers (19). For each maneuver we computed the relative venous return (RVR) using the following equation:

$$RVR = Ujv_M / Ujv_0 \quad (2)$$

Thus, for each maneuver in the first six series in Table 1 we obtained a value of relative venous return and a corresponding value of baseline-corrected airway pressure (ΔP_{ao}) from the same maneuver. By plotting RVR against ΔP_{ao} as shown in Fig. 4 we quantitatively illustrate Result 2: Venous return diminishes in a relatively linear manner as the level of airway pressure increases during a graded series of Valsalva maneuvers.

We next examined the degree to which a change in airway pressure is reflected in a change in right atrial pressure (RAP) during a Valsalva maneuver. The change in right atrial pressure from its resting, pre-maneuver value (ΔP_{ra}) and the corresponding change in airway pressure (ΔP_{ao}) are defined in Fig. 2. The comparison of these two quantities shown in Fig. 5 demonstrates Result 3: The relationship which we assumed in our previous work (19), that is, $\Delta P_{ra} = \Delta P_{ao}$, does not hold, but in fact $\Delta P_{ra} < \Delta P_{ao}$ during a Valsalva maneuver.

This result was obtained by externally compressing the lungs against a closed airway and is in contrast to the results obtained in series A.III. (last line of Table 1; lung inflations with chest and abdomen unrestrained). Except for the method of generating the stepwise changes in airway pressure, the conditions for series A.III. are essentially the same as for series A.I. However, as indicated in the second paragraph of this RESULTS section, the two techniques have entirely different effects on venous return; a level of airway pressure which practically stopped venous return for one heart cycle in series A.I. (17

cm H₂O; see Fig. 4) had almost no effect on venous return in series A.III. In addition, the two techniques yield entirely different results in terms of the relationship between the change in right atrial pressure (ΔP_{ra}) and the change in airway pressure (ΔP_{ao}) (Fig. 6A). The above observations are the basis for Result 4: Two possible methods for simulating Valsalva maneuvers in the anesthetized dog, chest compressions against a closed airway and lung inflations with an unrestrained chest, have qualitatively and quantitatively different effects on both right atrial pressure and venous return. The significance of this result in terms of the theoretical curves shown in Fig. 6B is discussed in Appendix B.

DISCUSSION

Noninvasive Measurement of Central Venous Pressure

Because of Result 1 and the data shown in Fig. 3, we must discount Webster's hypothesis that resting CVP can be estimated non-invasively by having the patient perform a graded series of Valsalva maneuvers while monitoring his venous return with a transcutaneous Doppler ultrasonic flowmeter (33,26). Wells and Ogden (35) arrived at essentially this same conclusion from measurements of both jugular and femoral venous flow in dogs, but the interpretation of their results is complicated by several factors. First, their data represent measurements over only a very narrow range of resting CVP (from -3 cm H₂O to +5 cm H₂O). Second, some variation is probably introduced into the results from their jugular venous flow measurements

because they apparently did not maintain a constant lung volume (and, therefore, constant relationship between airway pressure and intrathoracic pressure) before beginning each simulated Valsalva maneuver. The theoretical basis for this statement is found in our APPENDIX B. Finally, although their maneuvers while measuring femoral venous flow were apparently all begun from FRC, we are not certain that a consistent relationship holds between airway pressure and intra-abdominal pressure even under the conditions of lung compressions from a constant initial lung volume.

Relative Venous Return Curves and Venous Return Pressure

If Webster's central venous pressure hypothesis does not hold, how then do we interpret the quantitative decrease in venous return measured during a graded series of Valsalva maneuvers? First, we notice (by referring to Fig. 2) that the data represented in Fig. 4 were obtained by rapidly increasing right atrial pressure (RAP) from its resting level to a higher level and by determining relative venous return (RVR) in the first few seconds of the maneuver. Then the RAP was returned to its resting level, and the procedure was repeated again and again for different changes in RAP from the resting level. This is essentially the same technique suggested by Guyton as an alternative method for obtaining his venous return curves (12), even though he apparently has never specifically used a graded series of Valsalva maneuvers to determine a venous return curve. By plotting our values of RVR against their

corresponding values of right atrial pressure (Pra_M) (Fig. 7), we see that each of the regression lines represents the down-slope of the relative venous return curve for each of the first six series presented in Table 1. The curves are relative because venous return is normalized with respect to its value under the condition of resting (unperturbed) RAP. They are venous return curves because they are a plot of a measure of venous return (RVR) against right atrial pressure.

As with Guyton's "non-time dependent" venous return curves (12), the relative venous return curves shown in Fig. 7 (and the curves in Fig. 4 as well) may be thought of as "open loop transfer functions" in that they represent cardiovascular responses in the absence of compensatory reflexes. This is true because Guyton's curves are measured in animals which have been given total spinal anesthesia (12), and our curves are determined from values measured within the first 2 sec of the maneuver before the sympathetic cardiovascular reflexes begin to develop (29,12).

We now consider the significance of the above analogy between Guyton's venous return curves and our relative venous return curves obtained from graded series of Valsalva-like maneuvers. The intercept of Guyton's curve on the RAP axis corresponds to the value of the animal's mean systemic pressure (MSP), which is a measure of how well filled the systemic circulation is with respect to its capacitance (12). We propose, then, that the right atrial pressure intercept

($IPra_M$) of each of the regression lines in Fig. 7 similarly corresponds to the value of MSP for each of the six different conditions shown. This means that our Fig. 7 is analogous to Fig. 90 of Guyton's monograph (12), since both figures show how with increasing MSP, the venous return curve progressively shifts to the right with respect to the RAP axis.

In discussing the factors which determine venous return and, therefore, cardiac output, Guyton has emphasized what he calls the "pressure gradient for venous return," which is the difference between the value of mean systemic pressure and the value of right atrial pressure at any instant (13,12). From our data we should be able to estimate the pressure gradient for venous return (hereinafter referred to as venous return pressure or VRP) using the following equation:

$$VRP = IPra_M - \text{Pre-Maneuver CVP} \quad (3)$$

We perform the computations indicated in equation 3 for each of the six series conditions by taking $IPra_M$ values from the regression lines of Fig. 7 and by taking pre-maneuver CVP values from the last column of Table 1. The six calculated values of venous return pressure are shown in Fig. 8 plotted against their corresponding values of resting central venous pressure. From these data we conclude that venous return pressure is approximately constant (10.0 ± 1.8 (SD) cm H_2O , or 7.4 ± 1.4 (SD) mm Hg) throughout our experiment with this particular dog. Our calculated value for VRP corresponds well

with Guyton's average value of 6.9 ± 0.9 (SD) mm Hg for several hundred normal dogs (12).

Relationship Between Right Atrial Pressure and Airway Pressure

A possible application of our results for the study of human cardiovascular physiology would be to obtain relative venous return curves from consenting patients who are undergoing cardiac catheterization. Guyton's destructive technique (12) could never be applied to man, but mean systemic pressure and venous return pressure could be estimated in humans using our technique, which involves only a noninvasive flow (U_{jv}) measurement and an invasive pressure measurement (P_{ra}). Although we have discussed previously the noninvasive estimation of MSP and VRP (19), the possibility of this is complicated by our present result that the change in right atrial pressure is less than the change in airway pressure during a Valsalva maneuver (Result 3 and Fig. 5). We would hope that some consistently useful relationship obtains between these two quantities. The value of 0.48 for $\Delta P_{ra}/\Delta P_{ao}$ calculated from our time-domain data agrees well with the value of approximately 0.45 obtained by extrapolating the frequency-domain data of Wells and Ogden (34,35) for dogs to zero frequency. Using data reported in the literature (11) we calculate a mean value of $\Delta P_{ra}/\Delta P_{ao}$ of 0.63 during stage I of a 40 mm Hg Valsalva maneuver in man (six maneuvers in three different subjects), but it is not unreasonable to expect the value in humans to be different, on the average, from that in dogs.

To understand why $\Delta P_{ra}/\Delta P_{ao}$ during a Valsalva maneuver is less than unity, we consider this relationship to be composed of two functions:

$$\Delta P_{ra}/\Delta P_{ao} = (\Delta P_{ra}/\Delta P_{pl}) \times (\Delta P_{pl}/\Delta P_{ao}) \quad (4)$$

The basic question in interpreting this equation is: What is the pleural or intrathoracic pressure (P_{pl}) which actually affects the right atrium and central veins. The pressure in the esophagus (obtained via a balloon-tipped catheter) is commonly used as an indirect measure of local pleural-surface pressure, and the relationship between P_{es} and P_{ao} is well understood (20,22,1). However, even if ΔP_{es} can be accurately predicted by measuring ΔP_{ao} during a Valsalva maneuver, we are still uncertain of the relationship between P_{es} and the pressure around the right atrium and thoracic vena cavae. This is because of the largely unknown topography of pleural-surface pressures and pressure changes (1,20). In other words, although esophageal pressure may be equal to local intrathoracic pressure (P_{pl}), that particular pressure may not be equal to the intrapleural pressure surrounding the right atrium and central veins.

Even under the conditions when $\Delta P_{pl}/\Delta P_{ao}$ is unity and P_{pl} is the known extra-luminal central venous pressure, $\Delta P_{ra}/\Delta P_{pl}$ may be less than unity and not constant. This, of course, will make $\Delta P_{ra}/\Delta P_{ao} < 1$ through the relationship in equation 4. We have done some limited hybrid computer studies (Mastenbrook,

unpublished term project), using a circulatory model similar to that of Snyder and Rideout (30), to investigate the effects of stepwise changes in intrathoracic pressure on right atrial pressure. We found that there appear to be two competing mechanisms during a Valsalva maneuver: First, the change in P_{pl} tends to be transmitted through the compliant walls of the central veins and right atrium to the essentially incompressible blood inside, favoring the relationship $\Delta P_{ra}/\Delta P_{pl} = 1.0$. On the other hand, flow into the central veins and right atrium is diminished while outflow into the right ventricle continues, decreasing central venous volume and favoring the relationship $\Delta P_{ra}/\Delta P_{pl} < 1.0$. Other investigators have found that the right atrium makes a poor esophageal balloon in that it is difficult to accurately estimate changes in pleural pressure from changes in mean central venous pressure (24). Here we have confirmed this observation by examining the inverse problem, that of attempting to estimate changes in right atrial pressure from a known measure of changes in intrapleural pressure during a Valsalva maneuver (ΔP_{ao}).

Abdominal Compression as a Complicating Factor

Another problem in interpreting our results arises upon re-examination of Fig. 7 and Fig. 8: The conditions for series B.I. and B.II. result in different values for mean systemic pressure and venous return pressure even though the two series have approximately the same value of resting central venous pressure. In B.I. only the chest was compressed, and much of the force undoubtedly was directed through the diaphragm and into the

abdomen. Because of this, a greater external force may have been required to generate the same change in airway pressure than in B.II. where the abdomen also was compressed. If a non-uniform change in pleural pressure was generated, then it is conceivable that the pressure measured at the catheter tip was not the maximum pressure in the central veins. This would cause venous flow to cease at a lower measured right atrial pressure in series B.I. even though the actual pressure causing the flow to stop might be the same in both cases.

Since series B.II. involved compressing the abdomen, we also must consider the possibility that the mean systemic pressure was elevated by this procedure as suggested by Guyton (15,12). We do not know how much effect this phenomena has on pressure and flow in the jugular venous circuit early in each maneuver when we measure relative venous return, but what may be happening is a type of hepatojugular reflux (6). This consideration brings up the general question of how a Valsalva or Valsalva-like maneuver alters mean systemic pressure. Discussion of this problem and further speculation concerning possible clinical applications of the estimate of mean systemic pressure and venous return pressure must await the results of experiments wherein mean systemic pressure is measured directly, using Guyton's technique (12), and estimated indirectly, using our technique, in the same animal.

APPENDIX A.²List of abbreviations and symbols

CVP	- central venous pressure, synonymous with RAP
FRC	- functional residual capacity of the lungs
MSP	- mean systemic pressure
RAP	- right atrial pressure, synonymous with CVP
RVR	- relative venous return (ratio of U_{jv_M} to U_{jv_0})
VRP	- venous return pressure, synonymous with Guyton's term "pressure gradient for venous return"
PB	- local barometric pressure
Pes	- intraesophageal pressure
Δ Pes	- change in Pes from its pre-maneuver value at a given lung volume
P ℓ	- transpulmonary pressure
Δ P ℓ	- change in P ℓ from its pre-maneuver value at a given lung volume
Pp ℓ	- intrathoracic (pleural) pressure
Δ Pp ℓ	- change in Pp ℓ from its pre-maneuver value at a given lung volume
U_{jv}	- jugular venous blood flow velocity
U_{jv_0}	- value of U_{jv} just prior to a Valsalva maneuver
U_{jv_M}	- minimum value of U_{jv} during a Valsalva maneuver
Pra	- right atrial pressure
Pra ₀	- resting, pre-maneuver right atrial pressure (measured at the instant U_{jv_0} occurs)
Pra _M	- Pra measured at the instant U_{jv_M} occurs

- ΔPra - change in Pra from its resting, pre-maneuver value
(difference between Pra_M and Pra_O)
- $IPra_M$ - intercept of a relative venous return curve on the Pra_M axis
- Pao - airway pressure (measured at the opening of the airway)
- Pao_O - Pao measured at the instant Ujv_O occurs
- Pao_M - Pao measured at the instant Ujv_M occurs
- ΔPao - change in Pao from its pre-maneuver value (baseline-corrected Pao ; difference between Pao_M and Pao_O)
- Pao'_M - end-point Pao_M for a graded series of Valsalva maneuvers
(just sufficient that Ujv is zero for at least one heart cycle)
- Pao'_O - Pao_O for the maneuver which defines Pao'_M for each graded series of Valsalva maneuvers
- $\Delta Pao'$ - baseline-corrected end-point airway pressure (difference between Pao'_M and Pao'_O)
- Vl - lung volume
- ΔVl - change in lung volume from its initial, pre-maneuver value

APPENDIX B.

Theoretical relationships between airway pressure and intra-thoracic pressure during lung compressions and lung inflations.

The static compliance (volume-pressure) curve of the lung portion of the total respiratory system (lung plus chest wall) represents a plot of lung volume (V_l) versus the quantity

$$P_l = P_{ao} - P_{pl} \quad (5)$$

which is the pressure difference across the lung wall, or transpulmonary pressure (1). Now, if airway pressure (P_{ao}) and intra-thoracic pressure (P_{pl}) change during a lung compression or a lung inflation from a given lung volume, $\Delta P_{pl}/\Delta P_{ao}$ will be different from unity if and only if P_{ao} and P_{pl} change by different amounts. Equation 5 implies that

$$\Delta P_l = \Delta P_{ao} - \Delta P_{pl} \quad (6)$$

Therefore, if $\Delta P_{ao} \neq \Delta P_{pl}$, then there will be a change in transpulmonary pressure and a concomitant change in lung volume (in steady-state). The degree to which lung volume changes during the maneuver, then, determines the degree to which $\Delta P_{pl}/\Delta P_{ao}$ deviates from unity.

If we assume that there is no airflow during a lung compression maneuver against a closed airway, then the amount by which transpulmonary pressure changes from its initial value is determined by how much the volume of the lungs changes due to gas compression. This change in lung volume during a Valsalva-type maneuver may be calculated using Boyle's law (1):

$$Vl_2 = [(PB-47)Vl_1]/[(PB-47)+\Delta Pao] \quad (7)$$

$$\Delta Vl = Vl_2 - Vl_1 \quad (8)$$

where the subscripts 2 and 1 represent, respectively, final and initial values, PB is local barometric pressure, and 47 is the vapor pressure of water in mm Hg at 37°C. Assuming BP = 760 mm Hg, for a maximum ΔPao of 40 mm Hg (54 cm H₂O) we find that ΔVl is approximately 5% of the initial lung volume. This is negligible considering the accuracy of our measurements; therefore, we may consider $\Delta Ppl/\Delta Pao$ to be essentially unity during any one maneuver involving a compression of the lungs against a closed airway. The relationship between Ppl and Pao will then be

$$Ppl = Pao - Pl_1 \quad (9)$$

where Pl_1 is the initial transpulmonary pressure and is determined by the lung volume at the start of the maneuver (Vl_1). Equation 9 means that when estimating Ppl from the value of Pao measured during a lung compression maneuver, the results of two separate maneuvers may be compared in terms of the effect of a given value of airway pressure only if both maneuvers are begun from the same initial volume. This theoretical conclusion has been experimentally confirmed by others (9,21).

Lung compressions against a closed airway at functional residual capacity (FRC) and lung inflations from FRC may be compared quantitatively by using the lung mechanics model shown in Fig. 5 of Agostoni and Mead (1), which shows the static volume-pressure curves for the lung, the chest wall, and the

total respiratory system. We have chosen to begin all our experimental maneuvers and perform all our calculations from FRC because it represents the highly reproducible equilibrium volume of the respiratory system and is the only initial lung volume which has a relaxation pressure (initial P_{ao}) of exactly zero (atmospheric). From Fig. 5 of (1) we determine the change in intrathoracic pressure (from its value at FRC) for a given change in airway pressure (from atmospheric pressure). For lung compressions against a closed airway this is done as follows: For a given ΔP_{ao} and using FRC (in percent of vital capacity) for the value of V_{l_1} , we calculate V_{l_2} using equation 7. Reading across from this volume to the lung curve and down to the pressure scale we read the value of $P_{l_1} = P_{ao} - P_{pl}$ for that value of ΔP_{ao} . Since we know P_{l_1} and $P_{ao} = \Delta P_{ao} + 0$, we can solve for P_{pl} . Because of the small changes in lung volume for gas compressions (20), this calculation procedure gives essentially the same results as if we use equation 9 to calculate P_{pl} for any P_{ao} during a lung compression from FRC. The change in intrathoracic pressure is found by reading its initial value (at FRC) from the curve to be $-5 \text{ cm H}_2\text{O}$. Thus, using either of the above procedures for each value of airway pressure in $\text{cm H}_2\text{O}$, we can construct and calculate the corresponding value for the change in intrathoracic pressure in $\text{cm H}_2\text{O}$:

$$\Delta P_{pl} = P_{pl} - (-5) = P_{pl} + 5 \quad (10)$$

The same type of analysis for lung inflations beginning from FRC may be performed by realizing that for this one value of initial

lung volume the pressure axis of the compliance curve for the total respiratory system represents the change in airway pressure (ΔP_{ao}) and the pressure axis for the chest wall curve represents intrathoracic pressure (P_{pl}). From Fig. 5 of (1) we determine V_{l_2} for a given change in airway pressure by reading up from the value of ΔP_{ao} on the pressure axis to the respiratory system curve and horizontally across to the volume axis. We then find the corresponding value of P_{pl} by reading from this volume across to the chest wall curve and down to the pressure axis. In essence this procedure determines the value of intrathoracic pressure which exists under static conditions for a given value of relaxation pressure. Since the maneuver is begun from FRC, the initial value of intrathoracic pressure will again be $-5 \text{ cm H}_2\text{O}$, and ΔP_{pl} may be again calculated from equation 10.

The results of these theoretical interpolations are shown in Fig. 6B and may be compared to the experimental trends shown in Fig. 6A. Only a qualitative comparison is possible because the ventilatory compliance curves for the dog in our experiment are almost surely different from those in Fig. 5 of (1). But the results shown in Fig. 6 do explain an experimental finding common to both Wells and Ogden (35) and us (Result 4): Under no circumstances could venous return be stopped by inflating the lungs with external means with the abdomen and chest wall not restrained. This is because a lung inflation, even one approaching 100% vital capacity, does not generate a large enough change in intrathoracic pressure to impede venous return completely.

TEXT FOOTNOTES

¹The significance of this step of the procedure in terms of the importance of beginning all maneuvers from FRC is discussed in APPENDIX B.

²Where applicable, the symbols and definitions used herein conform to the standards recommended by the American Physiological Society (20,3).

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FIGURE LEGENDS

FIG. 1. Schematic of the experimental design. The cross-hatched regions on the ventral aspect of the supine dog indicate the positions of the investigator's hands when compressing the chest and/or abdomen. The physiologic reference level is explained in the text. The water manometers provide a visual indication of airway pressure and central venous pressure throughout the course of the experiment.

FIG. 2. Effect of a simulated Valsalva maneuver in the anesthetized dog. These records are from a single maneuver in the B.I. graded series of Valsalva maneuvers (see Table 1). The level of airway pressure generated in this maneuver is less than the end-point airway pressure for this series (i.e., $U_{jv_M} > 0$ for this maneuver). From the top the records are: A) differential output of the directional Doppler flowmeter; B) instantaneous output of the right atrial pressure transducer; C) instantaneous output of the airway pressure transducer. The Doppler flowmeter output is uncalibrated because only relative venous return can be measured with the transcutaneous probe. The mean levels of U_{jv} and P_{ra} (dashed lines) are time averages of their respective instantaneous waveforms. The O-subscripted quantities are measured just prior to the maneuver, and the M-subscripted quantities are measured at the instant U_{jv} reaches a minimum during the maneuver.

FIG. 3. Relationship between the airway pressure which just stops venous return and resting central venous pressure. Baseline-corrected end-point airway pressure ($\Delta P_{ao}'$) and pre-maneuver CVP are defined in the text. Each data point is from one of the first six graded series of Valsalva maneuvers described in Table 1.

FIG. 4. Effect of Valsalva-like maneuvers on relative venous return for the first six graded series of maneuvers described in Table 1. Relative venous return and baseline-corrected airway pressure (ΔP_{ao}) are defined in the text, and each regression line is a linear least-squares fit to the data.

FIG. 5. Relationship between the observed change in right atrial pressure and the corresponding change in airway pressure during graded series of simulated Valsalva maneuvers. Data points from the first six series of Table 1 are pooled in one plot. The solid line is the linear least-squares fit calculated to pass through the origin: $\Delta P_{ra} = (0.48)\Delta P_{ao}$; standard error of fit = 1.8 cm H₂O. The dashed line corresponds to the curve: $\Delta P_{ra} = (1.00)\Delta P_{ao}$.

FIG. 6. Comparison of two different techniques for generating changes in airway pressure. A) Experimental results. The conditions for series A.I. and series A.III. are described in Table 1. The dashed line is the linear least-squares fit calculated to pass through the origin. B) Theoretical prediction based on data taken from Fig. 5 of Agostoni and Mead (1) as discussed in APPENDIX B.

FIG. 7. Relative venous return curves obtained from each of the first six graded series of Valsalva maneuvers described in Table 1. \square :A.I.; \bullet :B.I.; \circ :B.II.; \blacksquare :C.II.; \circ :D.II.; \blacktriangle :E.II. Relative venous return (RVR) and right atrial pressure (Pra_M) are defined in the text. The right atrial pressure intercept (IPra_M) of each of the least-squares regression lines is the value of mean systemic pressure (MSP) for each of the six different conditions as described in the text.

FIG. 8. Relationship between estimated venous return pressure and resting central venous pressure. VRP is defined in equation 3 in the text, and pre-maneuver CVP is defined in Table 1. Each data point is from one of the first six conditions described in Table 1, and the dashed line is the level of the arithmetic mean of the six VRP values.

TABLE 1. Summary of experimental conditions

Series Designation	Method of Simulating Valsalva Maneuvers*	Number of Valsalva Maneuvers in Series, N	Pre-Maneuver Central Venous Pressure, cm H ₂ O [†]
A.I. [‡]	C	5	-0.6 ± 0.2
B.I. [§]	C	5	3.5 ± 0.4
B.II. [§]	C&A	7	3.8 ± 0.3
C.II.	C&A	6	8.6 ± 0.4
D.II.	C&A	8	15.8 ± 0.8
E.II.	C&A	7	21.9 ± 1.1
A.III. [‡]	LI	4	-0.9 ± 0.3

* C = Compression of chest only (abdomen unrestrained).

C&A = Compression of both chest and abdomen. LI = Lung inflation (chest and abdomen unrestrained).

[†] Mean of the N values of P_{ra_0} for each series ± standard deviation

[‡] A.I. and A.III. are considered to have essentially the same pre-maneuver CVP.

[§] B.I. and B.II. are considered to have essentially the same pre-maneuver CVP.

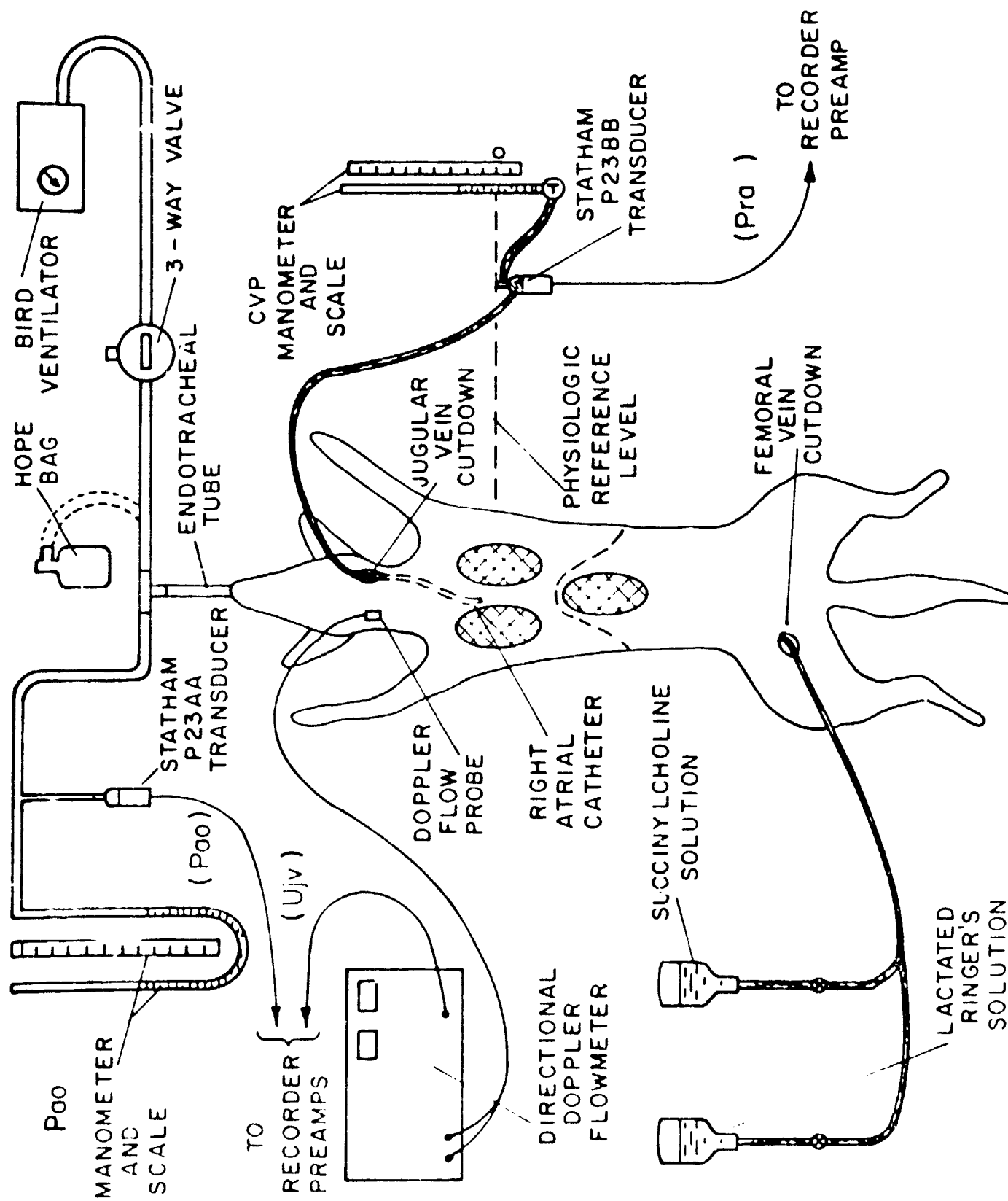


Figure 1

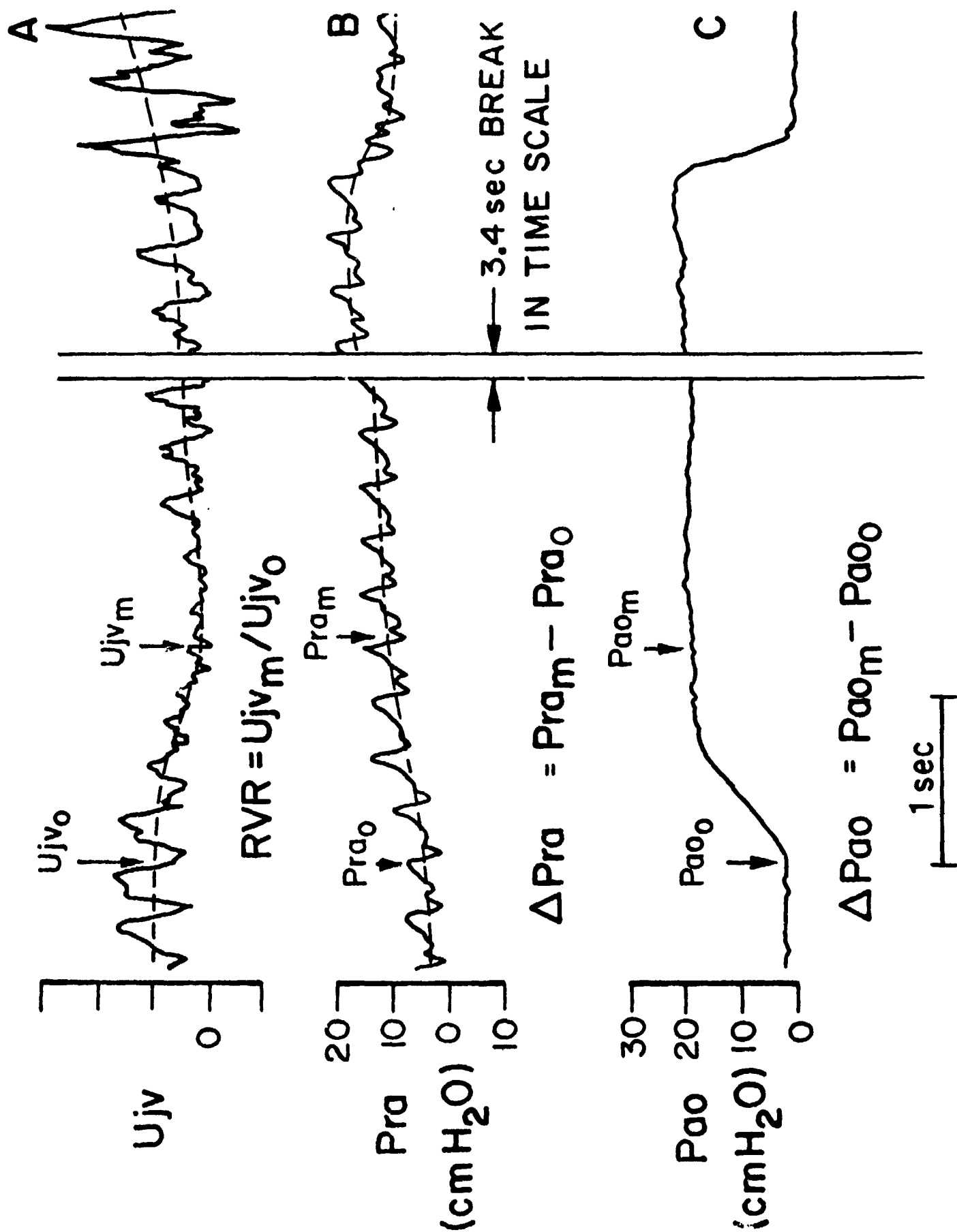


Figure 2

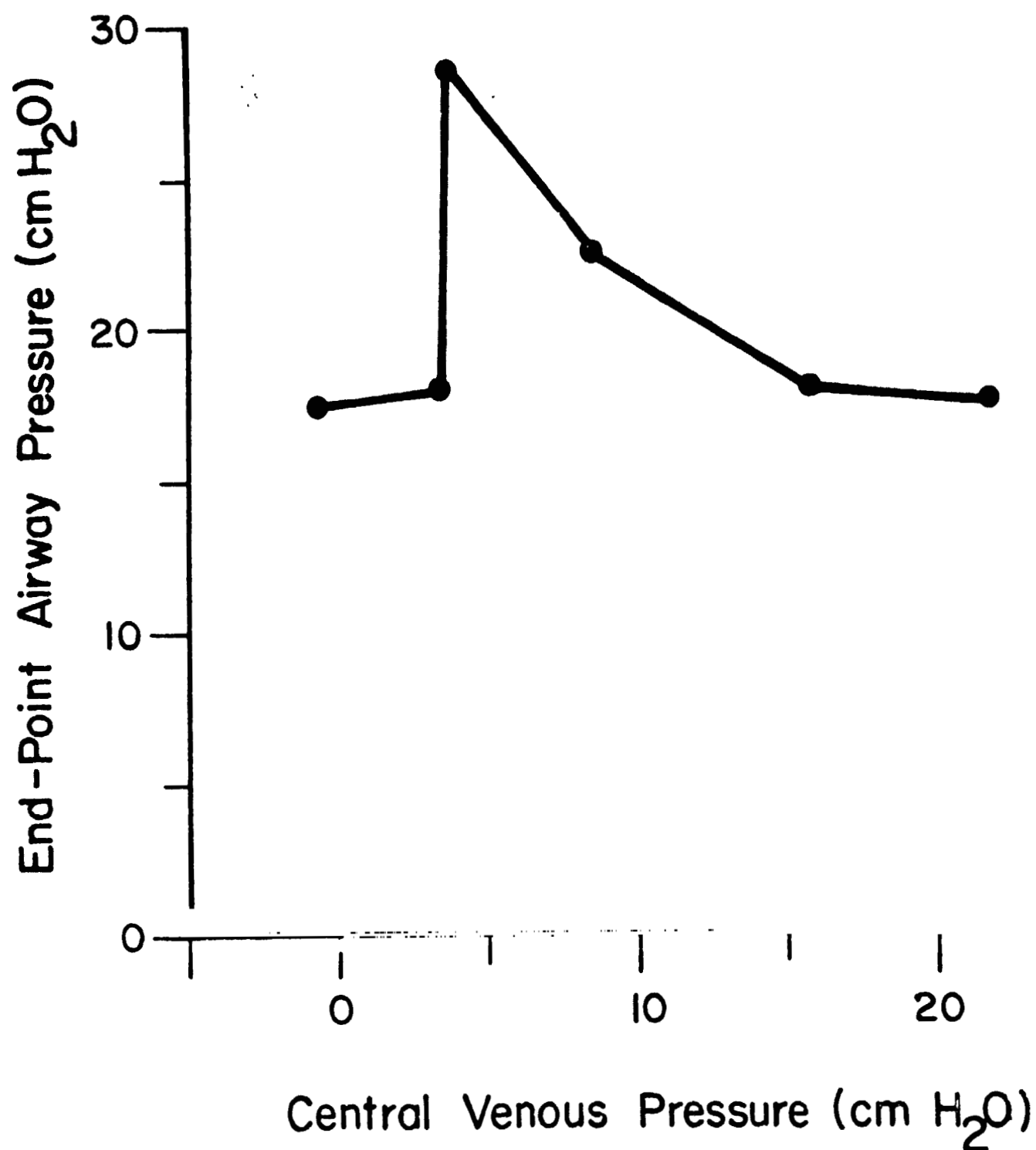


Figure 3

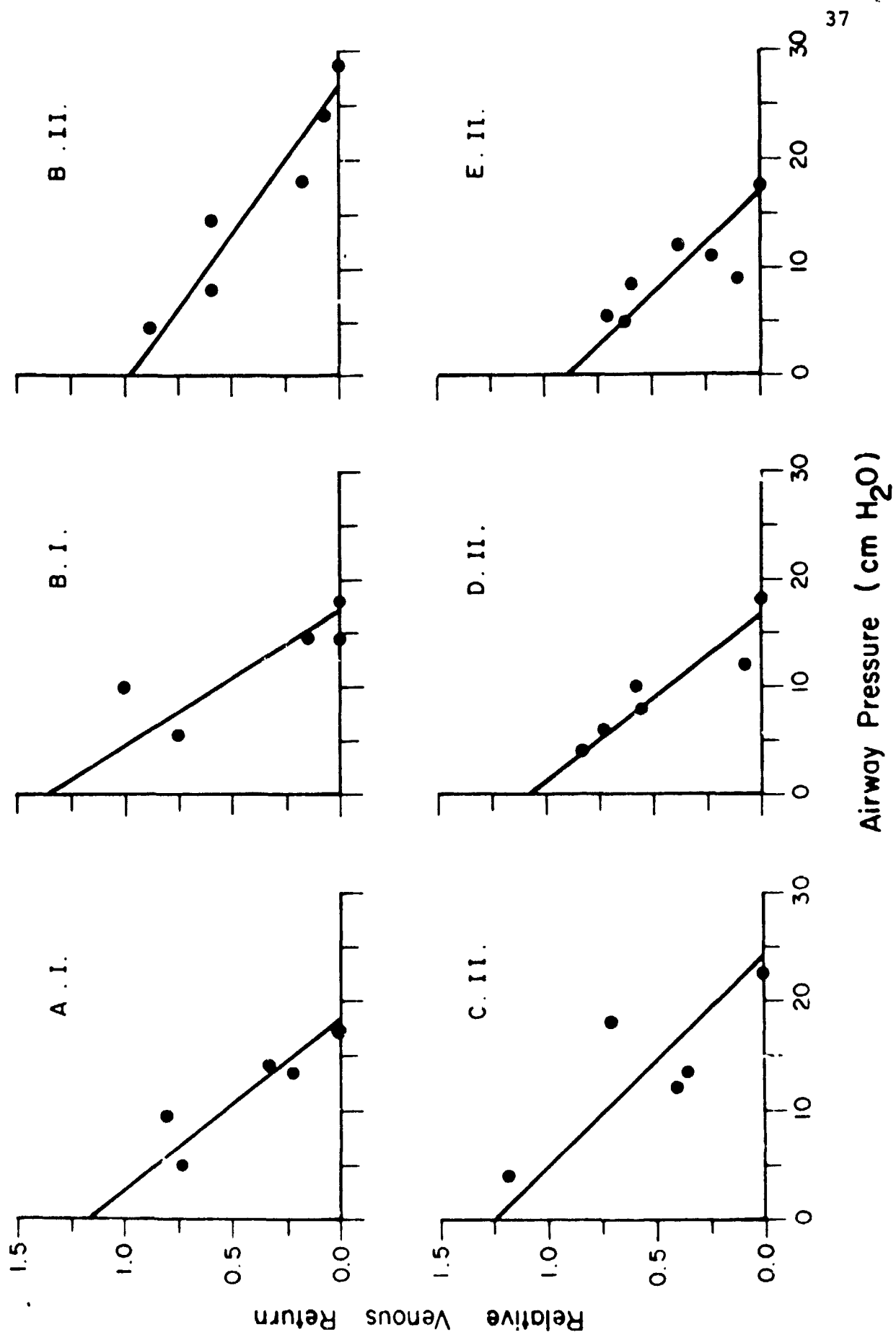


Figure 4

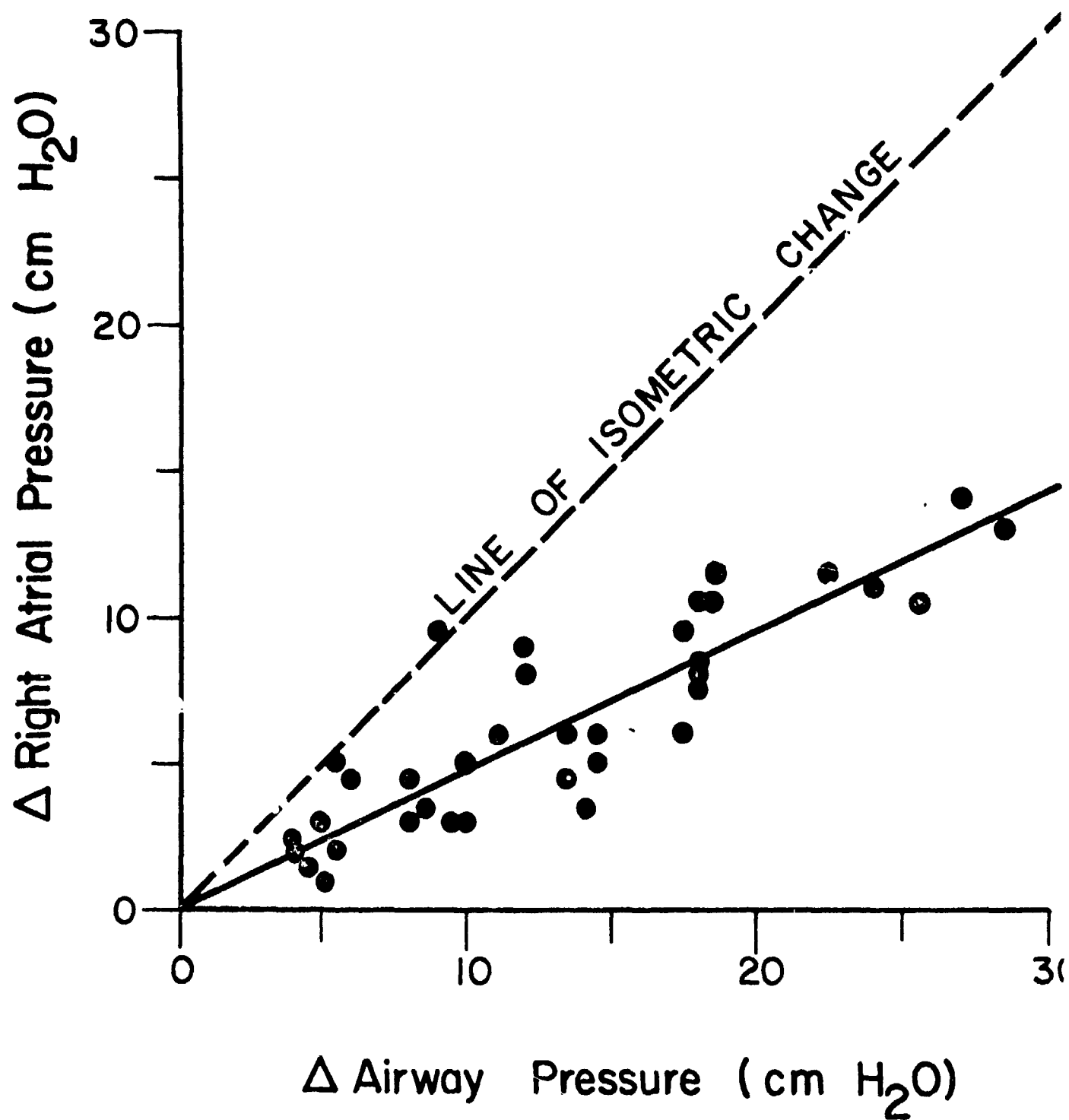


Figure 5

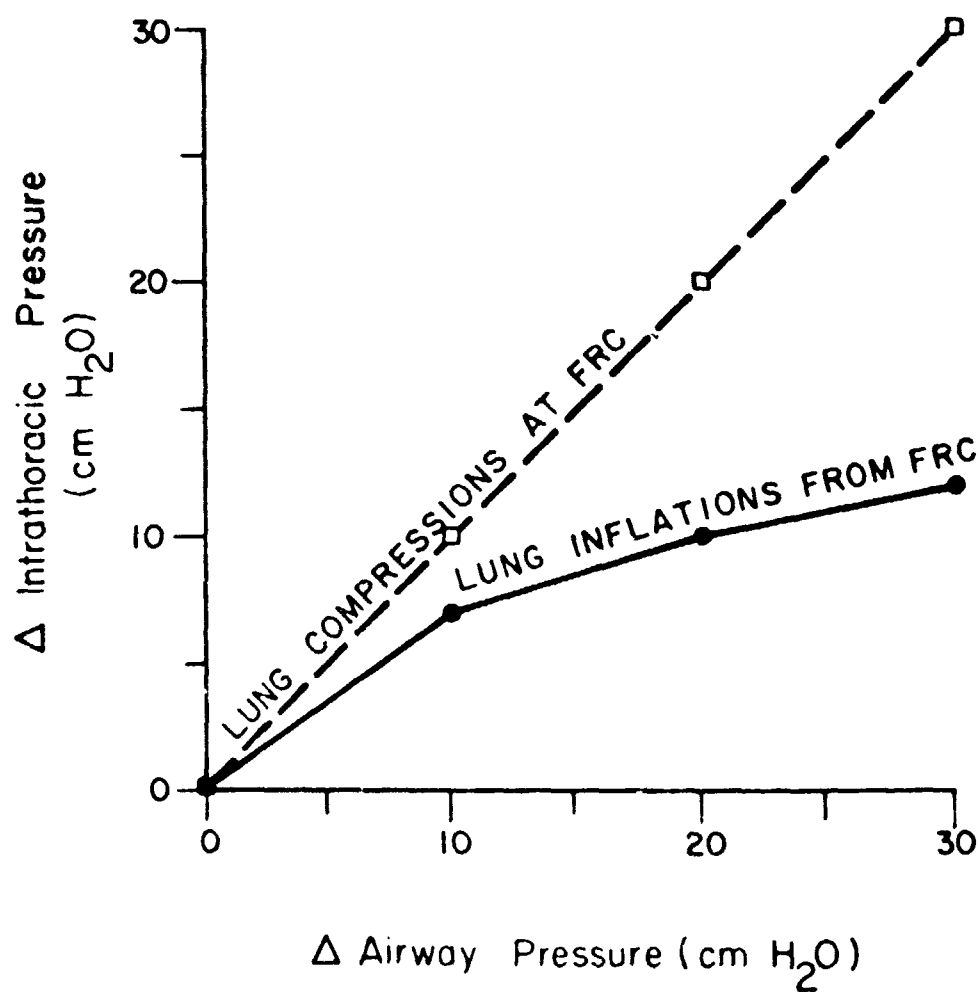
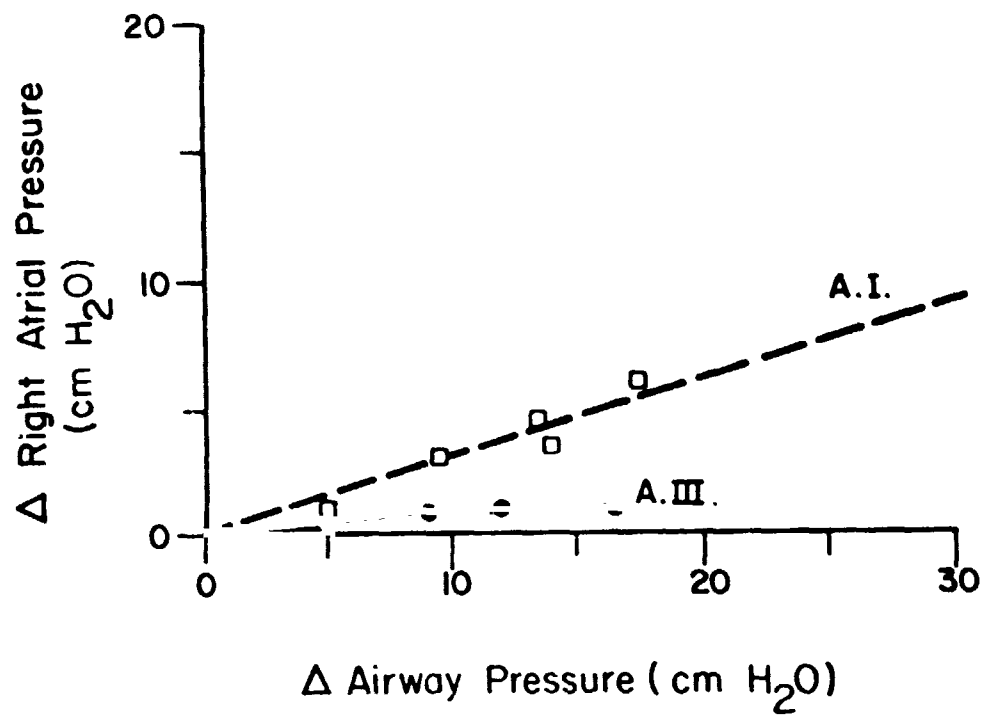


Figure 6

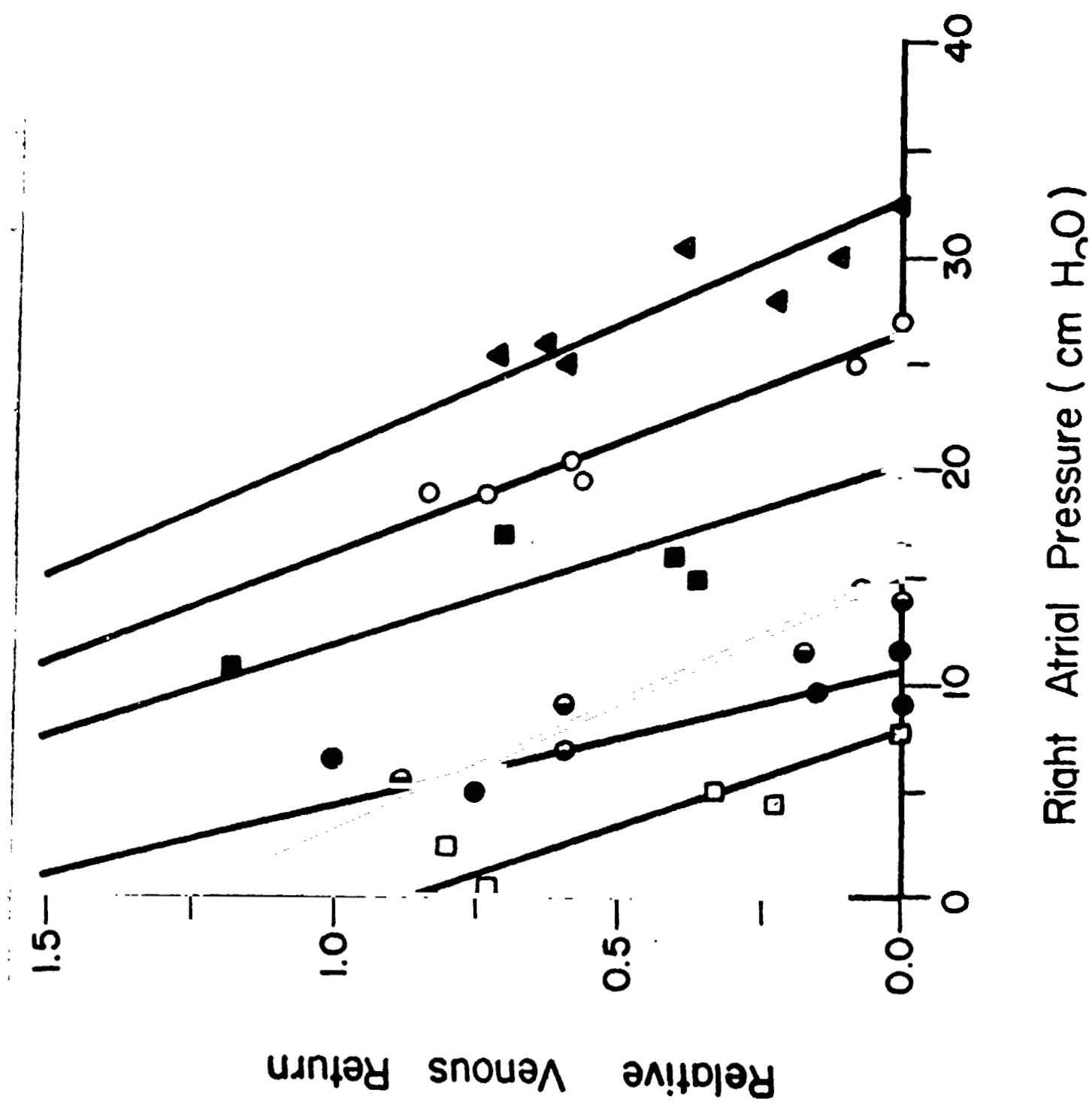


Figure 7

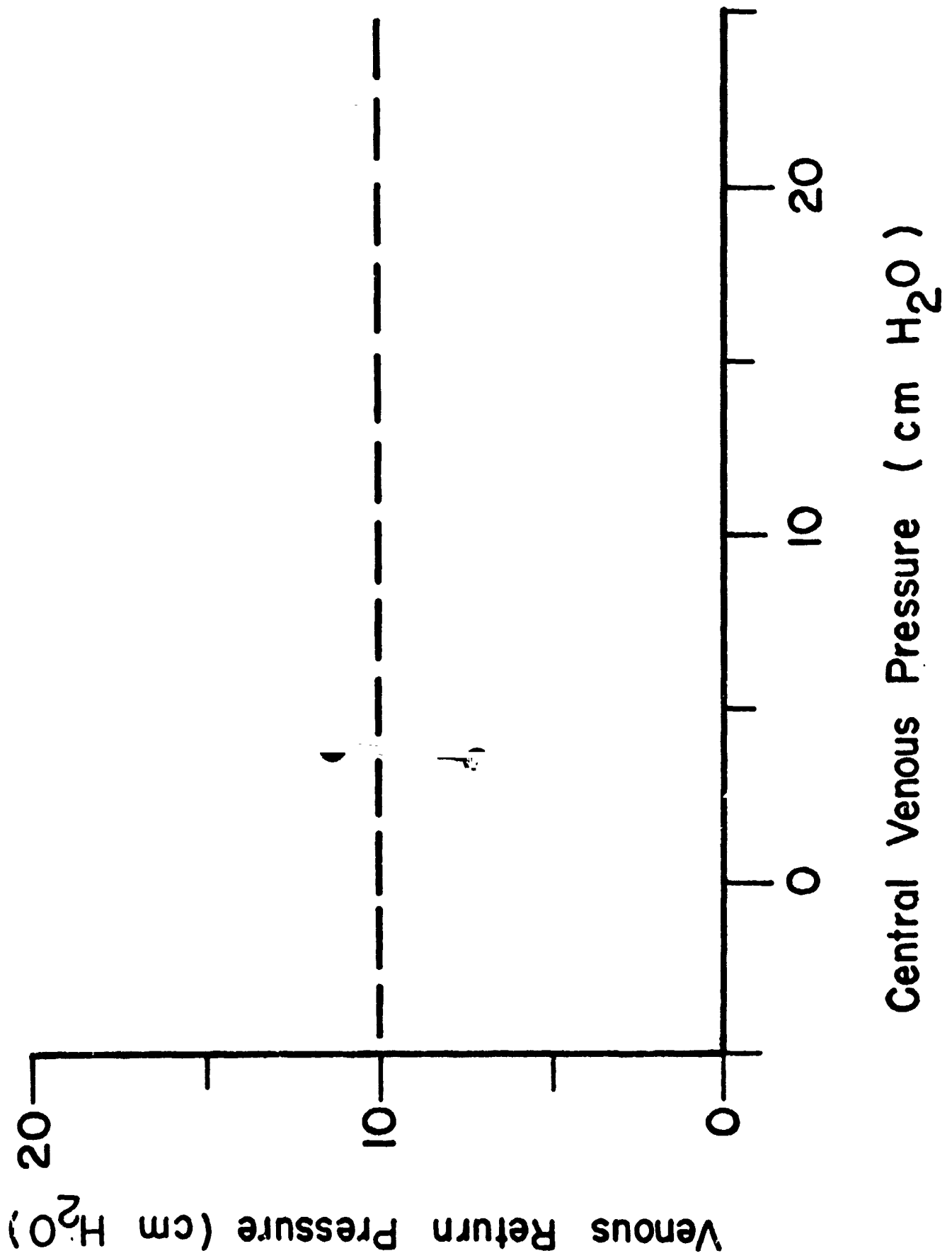


Figure 8